# Orthopedic Articular and Periarticular Joint Infections

Pim Jetanalin, MD[a,*], Yanint Raksadawan, MD[b], Pholaphat Charles Inboriboon, MD, MPH[c]

**KEYWORDS**

• Septic arthritis • Septic bursitis • Prosthetic joint infection • Monoarticular arthritis

**KEY POINTS**

- Septic arthritis is a "cannot miss" diagnosis of acute nontraumatic joint pain.
- Synovial fluid analysis must be interpreted in the context of a patient's risk factors.
- Diagnosing prosthetic joint infections requires a high index of suspicion given its varied presentation.
- All patients with suspected septic arthritis or prosthetic joint infections should be admitted and orthopedics surgery should be consulted.

## INTRODUCTION

Acute joint pain is commonly seen in the emergency department. The emergency physician must be adept in recognizing and treating the various causes of acute joint pain (**Box 1** differential diagnosis). Septic arthritis is an orthopedic emergency. If not promptly treated, it can result in serious complications including joint destruction and systemic infection that can lead to death.

The prevalence of septic arthritis is 2 to 10 in 100,000 in the general population.[1–4] Those in the extremes of ages: the first decade of life and the sixth decade of life are at the highest risk.[5–7] Patient-specific risk factors include injection drug use (IDU),[8] hemodialysis (HD),[8] immunocompromised states,[8] using immmunomodulators,[8] diabetes mellitus (DM),[5] and rheumatoid arthritis (RA; **Table 1** risk factors).[1,6] Joint-specific risk factors include pre-existing joint disease,[8] recent trauma,[5,6] previous articular procedures,[8] and periarticular soft tissue infection.[8] The most frequently identified pathogens are *Staphylococcus aureus* (54%–55%)[5,6,9] and *Streptococcus*

[a] Department Medicine, Division of Rheumatology, University of Illinois at College of Medicine, 818 South Wolcott Avenue, 6th Floor, MC 733, Chicago, IL 60612, USA; [b] Department of Medicine, Weiss Memorial Hospital, Medical Education, 4646 N. Marine Drive, Chicago, IL 60640, USA; [c] Department of Emergency Medicine, University of Illinois at College of Medicine, 808 South Wood Street MC 724, Chicago, IL, USA
* Corresponding author.
*E-mail address:* pimlin@uic.edu

Emerg Med Clin N Am 42 (2024) 249–265
https://doi.org/10.1016/j.emc.2024.01.002
0733-8627/24/Published by Elsevier Inc.
emed.theclinics.com

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SHOUT
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**Box 1**
**Differential diagnosis of acute joint pain**

Differential diagnosis for acute joint pain
- *Trauma*
  - Fracture
  - Hemarthrosis
  - Periarticular injury/internal derangement
- *Infectious*
  - Septic arthritis
  - Disseminated gonoccocal infection
  - Septic bursitis
  - Transient synovitis
  - Lyme arthritis
- *Crystalline diseases*
  - Gout
  - Calcium pyrophosphate dihydrate deposition disease
  - Basic calcium phosphate
  - Hydroxyapatite deposition disease
  - Calcium oxalate deposition disease
- *Inflammatory arthritis*
  - Atypical presentation/early presentation of polyarticular arthritis
  - Spondyloarthropathy
  - Juvenile idiopathic arthritis
  - Palindromic rheumatism
- *Other*
  - Pigmented villonodular synovitis
  - Lipoma arborescens
  - Synovial osteochondromatosis
  - Synovioma/sarcoma/metastatic disease
  - Neuropathic (Charcot joint)

spp (7.5%).[5,6] Methicillin-resistant *S aureus* (MRSA) accounts for 23% to 50% of isolates in contemporary case series.(Frazee 5)[90] Septic arthritis most commonly from hematogenous seeding but can also occur through local inoculation. Hence, a focused yet comprehensive history and physical examination are essential to diagnosing septic arthritis and the potential pathogen.

## CLINICAL PRESENTATION

Patients typically present with acute onset joint pain, swelling, and limited range of motion. Joint pain (85%) and swelling (78%) are the most sensitive symptoms.[10] Patients

**Table 1**
**Risk factors for septic arthritis**

| Patient-Specific Risk Factors | Local Risk Factors |
|---|---|
| • Immunocompromised states | • Pre-existing joint disease |
| • Using immunomodulators | • Previous articular procedures |
| • IDU | • Recent trauma |
| • HD | • Adjacent soft tissue infection |
| • DM | • Cutaneous ulcer |
| • RA | |
| • Extreme ages | |
| • Occupational risk factors | |

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may experience constitutional symptoms such as fever, sweats, and rigor. However, lack of these symptoms does not rule out septic arthritis as only 40% to 75% of patients have fever,[6,9–11] and 27% and 19% have sweats and rigor, respectively.[10]

The majority of patients with septic arthritis present with monoarticular arthritis. However, up to 22% have more than one joint involvement.[12] Therefore, septic arthritis cannot be excluded based on the number of joints affected.[12] Large joints are the most commonly affected, with knees representing more than 50% of cases.[5,6,8] Other commonly affected joints include the hip and shoulder. A history of IDU is a risk factor for septic arthritis involving fibrocartilaginous joints, such as the sacroiliac joint, sternoclavicular joint, or intervertebral disk.[13]

Risk factors for septic arthritis should be sought. In addition to IDU, these include pre-existing inflammatory joint diseases, such as RA and gout, a recent joint procedure and an immunosuppressed state, including use of immunosuppressive therapy and underlying systemic disease, including diabetes milletus, including diabetes. In patients with underlying inflammatory joint conditions, particularly RA, strongly consider septic arthritis if the patient develops new onset of acute arthritis in the setting of well-controlled disease, has a change in their typical flare pattern, or presents with constitutional symptoms. Maintain a high index of suspicion in patients who are immunocompromised, have prosthetic joints, or are infected with atypical organisms because they may present with a more insidious course.

## *Special Populations*

### *Immunocompromised hosts*

Immunocompromised hosts, such as patients with acquired immunodeficiency syndrome and patients who require immunosuppressive medications may present atypically with septic arthritis. Patients may have a chronic indolent course with pain for months rather than an acute onset.[14,15] Some may present without the typical physical examination findings such as joint swelling and only present with pain and range of motion (ROM) limitation.[16] Some may present with an infection due to more than one pathogen.[17]

These patients are also prone to infections due to rare pathogens. Septic arthritis may be the first manifestation of a disseminated infection with a rare pathogen, such as *Listeria monocytogenes*,[1] *Mycobacterium avium* complex,[18] *Histoplasma capsulatum*,[19] *Aspergillus fumigatus*,[20] *Brucella* spp,[21] *Salmonella enteritidis*,[22] *Mycoplasma pneumoniae*,[23] or *Blastomyces dermatitidis*[14] (**Fig. 1**). This wide variety of potential pathogens underscores the importance of obtaining synovial fluid cultures.

### *Underlying rheumatologic disease*

Patients with certain underlying rheumatologic conditions also have an increased risk for septic arthritis, both because of immunosuppressive medications and pre-existing joint pathology. Other risk factors that increase risk in this patient population include older age, male sex, HTN, DM, and seropositive RA diagnosis.[24] Tumor necrosis factor inhibitors, which are one of the most widely used biologics in RA and spondyloarthropathies, have been shown to increase the risk of septic arthritis.[24,25] In contrast to the general population, a retrospective study found that the most common pathogen for septic arthritis in patients with SLE was *Salmonella*, followed by *Mycobacterium*, and then *S aureus*.[26] High-dose steroids in patients with systemic lupus erythematosus were found to be a predisposing factor for salmonella infection.[27]

### *Physical examination*

The affected joint should be exposed and assessed. Generalized tenderness, erythema, warmth, and pain with both passive and active range of motion should increase

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**Fig. 1.** A 64 year old man with a history liver transplant on immunosuppressive therapy presented with subacute onset of left knee pain, swelling, and decreased ROM. Patient was found to have disseminated blastomycosis.

your suspicion for septic arthritis. On the contrary, in periarticular pathology (ie, tendinitis, bursitis, and periarticular gout), range of motion is relatively preserved and pain only occurs during active ROM. Swelling, point of tenderness, and erythema are typically more localized.[28]

In addition to a focused joint examination, a focused physical examination including a skin examination looking for lesions, pustules, wounds, cellulitis, and other skin or soft tissue infections should be performed. In patients with fibrocartilaginous joint infections, patients should be examined for other sources of infection, such as skin abscess and infectious endocarditis.

***Diagnostic tests***

Diagnosis can be challenging due to lack of both reliable and specific laboratory and imaging findings. Patients may have leukocytosis or elevated acute phase reactants. C-reactive protein (CRP) is of particular diagnostic value because it is more sensitive than the white blood cell (WBC) count or erythrocyte sedimentation rate (ESR).[6,9] Only two-thirds of patients with septic arthritis have a leukocytosis greater than 11,000 cells/mm$^3$.[6,11] It is important to note that these laboratory values are nonspecific and can be seen in other types of inflammatory arthritis as well as in nonjoint infections such as osteomyelitis and bursitis.

In early septic arthritis, plain radiographs may be normal or it may reveal periarticular osteopenia, soft tissue swelling, and effusions.[29,30] In advanced disease, there can be joint space narrowing, nonspecific erosion, and destruction.[29,31] CT and MRI are more sensitive in detecting soft tissue changes[30] and can help identify which anatomic compartment is affected.[32] MRI is the most sensitive modality for detecting inflammatory arthritis, including septic arthritis. Early in the disease process, joint effusion and

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synovial inflammation is seen. This is followed by early marrow signal abnormality and subsequent cartilage loss and subchondral bone erosion.[33] Ultrasound can help detect joint effusions that may be missed by plain radiographs.[34] In the case of hip ultrasounds, the lack of an effusion has a high negative predictive value for septic arthritis.[35] Additionally, it can be used as a point-of-care tool to guide joint aspiration.[36,37]

## ARTHROCENTESIS AND SYNOVIAL FLUID ANALYSIS

Arthrocentesis should be performed in all patients with suspected septic arthritis. It can be performed using anatomic landmarks or with the assistance of imaging modalities such as ultrasound and fluoroscopy. Needle aspiration through overlying cellulitis is generally contraindicated; however, some advocate going through infected skin when it cannot be avoided given the risk of delayed diagnosis and treatment.[10] In cases in which arthrocentesis does not yield an adequate amount of fluid, sterile saline can be used to irrigate the joint to obtain fluid for gram stain and culture analysis.[38]

Synovial fluid analysis is an essential diagnostic test in the evaluation of possible septic arthritis. Synovial fluid should be sent for cell count with differential, gram stain, culture, and crystal analysis. Gram stain is positive in about half of patients with septic arthritis. Cultures are considered the gold standard but are only positive in 71% of patients with native joints infections and in 82% of patients with prosthetic joints infections.[11] Given the relatively low yield of gram stains and that culture results are not immediately available, synovial cell count is essential for determining the likelihood of septic arthritis. The likelihood ratio of having septic arthritis increases with an increasing synovial fluid WBC count[10] (**Tables 2** and **3**). Patients with synovial WBC count greater than 50,000 cells/mm$^3$ should be treated empirically for septic arthritis. Immunocompromised patients may have a lower than expected synovial WBC count.[39] It is important to note that crystalline disease (gout and calcium pyrophosphate disease) with or without acute flare can coexist with septic arthritis. Therefore, the presence of crystals does not rule out septic arthritis.[40]

### *Treatment*

Patients with suspected septic arthritis should be admitted and orthopedics should be consulted. Immediate orthopedic consult is particularly crucial in suspected prosthetic joint infection (PJI). In clinically uncertain cases, patients should be admitted for close monitoring and further evaluation while awaiting culture results.

Treatment of septic arthritis includes both antibiotics and removal of infected synovial fluid. Empiric antibiotic selection is guided by the results of gram stain, patient risk

**Table 2**
**Synovial cell count and likelihood of septic arthritis**

| Synovial Fluid WBC Count | Likelihood Ratio |
|---|---|
| WBC <25,000 | 0.32 |
| WBC >25,000 | 2.9 |
| WBC >50,000 | 7.7 |
| WBC >100,000 | 28 |
| Synovial PMN >90% | 3.4 |
| Synovial PMN <90% | 0.34 |

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**Table 3**
**Diagnostic characteristics of synovial fluid**

| Characteristics of Synovial Fluid | Normal | Noninflammatory | Bacterial Septic Arthritis | Disseminated Gonococcal Infection | PJI |
|---|---|---|---|---|---|
| Appearance | Transparent | Transparent | Cloudy | Cloudy | — |
| Viscosity | High | High | Variable | Variable | — |
| WBC count (cells/mm$^3$) | <200 | 200–2000 | 2000–>50,000 | 13,000–100,000 | >10,000 in early onset<br>>3000 in late onset |
| Neutrophils (%) | <10 | <10 | >90 | 90 | >90 in acute<br>>80 in chronic |
| Gram stain | Negative | Negative | Positive | <50% showing intracellular and extracellular gram-negative diplococci | Positive |
| Culture | Negative | Negative | Positive | <50% of cases | Positive |

factors, and the list of common causative pathogens (**Table 4**). For patients in communities with high MRSA prevalence, known MRSA colonization, or other risk factors for MRSA infection, vancomycin is recommended.[39,41] Cefazolin is recommended for patients at low risk for MRSA.[39] Immunocompromised, elderly, or IDU populations are at risk for gram-negative infections, particularly *Pseudomonas aeruginosa*, and addition of an antipseudomonal beta-lactam such as cefepime or piperacillin-tazobactam should be considered.[39,41] There is no consensus on the duration of antibiotic therapy, with recommendations ranging from 2 to 6 weeks, including step-down oral antibiotics.[39] Clinical improvement and acute phase reactants can be used to assess response to treatment and duration of therapy.[42]

In addition to establishing the diagnosis of septic arthritis, arthrocentesis aids in treatment, decreasing the organism load, inflammatory mediators,[43] and intra-articular pressures that can lead to localized joint destruction. Patients can be treated medically with serial arthrocentesis or surgically with arthroscopy or open arthrotomy. Though there is a higher rate of treatment failure in patients treated with serial arthrocentesis, patients that undergo surgery tend to have longer hospital stays, are less likely to achieve full recovery, and are more likely to have residual joint deformities.[44,45] Patients with underlying joint disease, abscess formation, positive synovial culture, hip joint involvement, or osteomyelitis are more likely to require surgical treatment.[46]

### *Prosthetic Joint Infection*

PJI is a feared complication of joint replacement that causes significant morbidity and mortality and is the most common cause of total knee arthroplasty failure.[47] PJI has an incident rate of 1.4% to 1.7%.[48,49] The prevalence of PJI has increased 2.5-fold from 2005 to 2013 in primary knee replacements, and 7.5-fold in revision knee replacements.[50] Risk factors for developing PJI include male sex,[48] young age,[48] type 2 DM,[48] posttraumatic arthritis,[48] patellar resurfacing,[48] discharge to a convalescent home,[48] malnutrition,[51] morbid obesity,[51] immunocompromised state,[51] preoperative anemia,[52] and *S aureus* colonization.[51]

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**Table 4**
**Empiric antibiotic regimens for patients with suspected septic arthritis**

| Synovial Fluid Gram Stain | Antibiotic Coverage | Dose |
|---|---|---|
| Gram-positive cocci in clusters (presumed *Staphylococcus*) | Cefazolin | 2 g IV every 8 h |
| Gram-positive cocci in clusters (presumed MRSA in high-prevalence area) | Vancomycin | 1 g IV every 12 h |
| Gram-positive cocci in chains (presumed *Streptococcus*) | Cefazolin | 2 g IV every 8 h |
| Gram-negative diplococci (presumed gonococcus) or clinical presentation suggestive of disseminated gonoccal infection | Ceftriaxone<br>+Azithromycin | 1 g IV every 24 h<br>1 g PO (single dose) |
| Gram-negative bacilli | Cefepime or Piperacillin-tazobactam | 2 g IV every 8 h or 4.5 g IV every 6 h |

In the absence of an organism on gram stain, consider vancomycin 1 g IV every 12 h (cefazolin may be used in patients at low risk for MRSA in a low prevalence area). An antipseudomonal beta-lactam, such as cefepime or piperacillin-tazobactam, should be added in the elderly, immunocompromised, critically ill, or intravenous drug users.

John J Ross. Septic Arthritis of Native Joints. Infect Dis Clin North Am. 2017 Jun;31(2):203-218.[39]

The most common pathogens are *S aureus* and coagulase-negative *Staphylococcus*.[52–54] *Candida* infection has been reported in patients with systemic autoimmune diseases and in those who are immunocompromised or have chronic diseases such as renal disease.[55]

PJI can be divided into 3 categories, by time of onset: early (<3 months), delayed (3–12 months), and late (>12 months; **Table 5**). Early infection is typically caused by relatively virulent pathogens introduced during surgery, including *S aureus* and gram-negative bacteria. Delayed infection is usually caused by less virulent pathogens introduced during surgery. Late PJI often occurs through hematogenous seeding of the prosthetic joint from a remote source of infection.[53]

In addition to the classic symptoms of septic arthritis, features that are highly suspicious for PJI include persistent drainage from operative wound, the presence of a sinus tract, acute onset of pain over a prosthetic joint that was previously asymptomatic, and persistent pain after joint replacement without a pain-free period.[56] An orthopedic specialist should be consulted immediately in patients with suspected PJI.

Plain x-rays of the prosthetic joint may be of limited diagnostic utility in suspected PJI because radiographic findings may lag by 1 to 2 weeks from the onset of clinical findings. MRI is a more sensitive imaging modality, but it may be limited by metal artifact from the prosthetic joint.[57]

Synovial WBC elevation is not able to differentiate between infectious and other inflammatory arthropathies.[57,58] Preoperative synovial aspiration culture (bedside arthrocentesis of the infected joint) is positive in only 45.2% of knee PJI and 44.4% of hip PJI.[59] It cannot be used alone to rule out PJI because the false-negative rate is as high as 20.7% in knee PJI and 15% in hip PJI.[59]

The 2018 definition of periprosthetic hip and knee infection criteria has a sensitivity and specificity of 97.7% and 99.5% (**Fig. 2**: PJI Diagnostic Criteria).[60] PJI is diagnosed

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**Table 5**
**Characteristic of prosthetic joint infection based upon onset**

| Time to Infection After Implantation | Early Onset | Delayed Onset (3–12 mo) | Late Onset |
|---|---|---|---|
| Synovial fluid | | | |
| WBC (cells/μL) | >10,000 | >3000 | >3000 |
| Neutrophils (%) | >90 | >80 | >80 |
| Clinical symptoms | Wound drainage, erythema, joint pain, fever | Possible sinus tract formation, subacute joint pain | Systemic symptoms with bacteremia, pain |
| Serum ESR (mm/h) | Not useful | >30 | >30 |
| Serum CRP (mg/L) | >100 | >10 | >10 |
| Common pathogens | Virulent organisms:<br>*S aureus*<br>Aerobic gram-negative bacilli<br>Polymicrobial<br>Anaerobic | Low virulence organisms:<br>Coagulase-negative staphylococci<br>*Enterococcus*, *Cutibacterium* | *S aureus*<br>β-Hemolytic streptococci<br>Gram-negative bacilli |

*Modified from* Beam E, Osmon D. Prosthetic Joint Infection Update. Infect Dis Clin North Am. 2018;32(4):843-859. https://doi.org/10.1016/j.idc.2018.06.005.[53]

when 1 of 2 major criteria is present, or scoring 6 or more based on minor criteria. Major criteria are 2 positive cultures growing the same organism and the presence of a sinus tract with evidence of communication to the joint, including visualization of the prosthesis. Minor criteria are based on serum and synovial fluid testing. Serum ESR, CRP, and d-dimer should be ordered and synovial cell count with differential and synovial CRP and alpha-defensin should be obtained. Synovial fluid alpha-defensin has high sensitivity and specificity for PJI.[60,61]

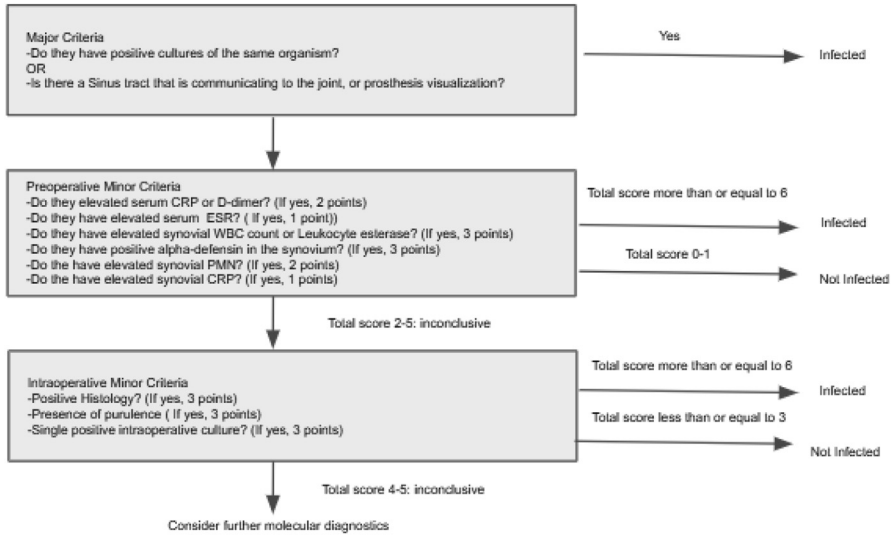
Major Criteria
-Do they have positive cultures of the same organism?
OR
-Is there a Sinus tract that is communicating to the joint, or prosthesis visualization?
Yes
Infected
Preoperative Minor Criteria
-Do they elevated serum CRP or D-dimer? (If yes, 2 points)
-Do they have elevated serum ESR? ( If yes, 1 point))
-Do they have elevated synovial WBC count or Leukocyte esterase? (If yes, 3 points)
-Do they have positive alpha-defensin in the synovium? (If yes, 3 points)
-Do the have elevated synovial PMN? (If yes, 2 points)
-Do the have elevated synovial CRP? (If yes, 1 points)
Total score more than or equal to 6
Infected
Total score 0-1
Not Infected
Total score 2-5: inconclusive
Intraoperative Minor Criteria
-Positive Histology? (If yes, 3 points)
-Presence of purulence ( If yes, 3 points)
-Single positive intraoperative culture? (If yes, 3 points)
Total score more than or equal to 6
Infected
Total score less than or equal to 3
Not Infected
Total score 4-5: inconclusive
Consider further molecular diagnostics

**Fig. 2.** A scoring-based definition for periprosthetic joint infection (PJI).

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When patients are clinically stable, the The Infectious Diseases Society of America (IDSA) recommends withholding antimicrobial therapy for at least 2 weeks to increase the likelihood of positive culture.[62] Definitive PJI treatment requires antibiotic therapy plus surgical debridement or joint replacement. A 12 week course of oral antibiotic therapy decreases the risk of treatment failure.[63,64]

### *Gonococcal Arthritis (Disseminated Gonococcal Infection)*

Gonococcal arthritis is seen predominantly in sexually active young adults with a 3:1 female predominance.[65] It is postulated that localized infections in female individuals are typically asymptomatic, which leads to delayed diagnosis and progression to disseminated infection.[66] Gonococcal arthritis classically presents as a triad of migratory polyarthritis, tenosynovitis, and skin lesions. However, only about a quarter of patients present with all of these symptoms.[67] Patients may present with migratory polyarthralgias or polyarthritis. True arthritis is only present in 50% of patients, and commonly affected joints include the knee, ankle, elbow, and wrist. Tenosynovitis is typically noted in the wrist, ankles, finger, or toe joints. Dermatitis is present in about 75% of patients and is often present on the extremities as nonpainful nonpruritic macules, papules, pustules, or vesicles (**Fig. 3**). Lesions are rarely noted on the face.[65,66,68]

Synovial fluid WBC count is typically lower than in classic septic arthritis, ranging from 13,000 to 100,000 with neutrophil predominance.[67] *Neisseria gonorrhoeae* is identified by gram stain in about half of confirmed cases of gonoccocal arthritis, and synovial cultures are positive in less than half of cases.[65] Synovial nucleic acid amplification tests (NAAT) can detect infection in synovial fluid, although this test is neither widely available nor standardized.[65] In addition to the traditional septic arthritis evaluation, a comprehensive sexual history should be obtained in patients with suspected gonoccocal arthritis, with cultures or NAAT of multiple mucosal sites (oropharyngeal, cervical, urethral, and anorectal) recommended. Upward of 80% of patients with gonococcal arthritis are diagnosed based on clinical presentation and nonsynovial, mucosal NAAT, or cultures.[66] Blood cultures are positive in less than 50%, and cultures of skin lesions have a very low yield.

With early recognition and prompt treatment, 95% of patients will have a full recovery.[69] The Centers for Disease Control and Prevention recommends hospitalization and infectious disease consultation for the treatment of gonoccocal arthritis. The initial recommended treatment is ceftriaxone 1 g intravenously (IV) or intramuscularly every 24 hours. An

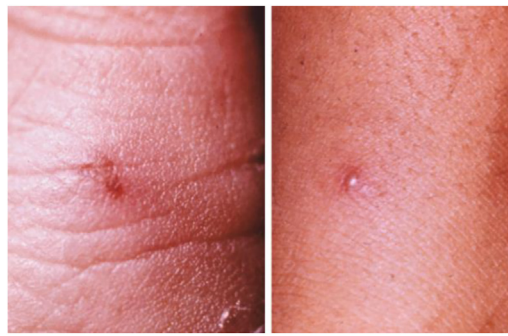

**Fig. 3.** Skin lesions in disseminated gonococcal infection. (Joshua Landy, Dennis Djogovic, Wendy Sligl, Gonococcal septic shock, acute respiratory distress syndrome, and multisystem organ failure: a case report, International Journal of Infectious Diseases, 14, Suppl 3, 2010, e239-e241, https://doi.org/10.1016/j.ijid.2009.08.021.)

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alternative option is either cefotaxime 1 g IV every 8 hours or ceftizoxime 1 g every 8 hours. Patients generally should be tested and empirically treated for chlamydial infection also, and should undergo testing for other sexually transmitted infections.[70]

### *Septic Bursitis*

Septic bursitis is frequently due to direct trauma rather than hematogenous infection. The olecranon and prepatellar bursa are the most commonly affected sites because of their location and frequency of adjacent trauma.[71] Deep septic bursitis is uncommon and diagnosis is often delayed.[72] The annual incidence of acute olecranon and prepatellar bursitis is 10 in 100,000, with one-third of the cases being septic.[73] Cases are predominantly male individuals (80%–89%),[74–77] with a mean age of onset reported at 49.7 to 57 years of age.[74–78]

The most prominent risk factor for septic bursitis is repetitive motion and trauma,[71,77,78] which is present in 47% to 77% of cases.[79] There is a well-known association between occupation and certain types of bursitis. For example, occupations that involve constant kneeling are associated with prepatellar bursitis, sometime referred to as "housemaid's knee." This condition is also seen in Carpet layers. Janitors and laborers are more likely to experience olecranon bursitis, while automobile mechanics and carpenters have an increased risk of developing both olecranon and prepatellar bursitis.[71] Other risk factors for septic bursitis include underlying comorbid conditions such as HD,[71] chronic obstructive pulmonary disease (COPD),[71] DM,[71] chronic alcohol use,[71,77] previous traumatic bursitis,[71] RA,[71] gout,[71] and steroid use.[71]

*S aureus*, including MRSA, is the predominant pathogen[74,78,80–82] accounting for 87.5% to 94.4%[77,82] of cases. Less common potential pathogens include skin flora such as *S epidermidis* and streptococcal species.[83] Septic bursitis due to exotic pathogens has been described in immunocompromised individuals, including *Mycobacterium kansasii*,[84] *Candida* spp,[81,85] *Salmonella*,[86] and *Cryptococcus neoformans*.[81]

#### *Clinical presentation*

Patients typically present with localized tenderness, erythema, and warmth over the bursa area (**Fig. 4**). In contrast to septic arthritis, the joint motion is usually preserved.[71] It may be difficult to determine whether bursitis is septic or aseptic. In comparison to aseptic bursitis, septic bursitis has an increased frequency of tenderness (88 vs 36%), erythema (83 vs 27%), warmth (84 vs 56%), trauma (50 vs 25%), and fever (38 vs 0%; **Table 6**).[79]

**Fig. 4.** Septic olecranon bursitis note the localized erythema and swelling over the olecranon bursa. (Danielle Reilly, Srinath Kamineni, Olecranon bursitis, Journal of Shoulder and Elbow Surgery, 25 (1), 2016, 158-167, https://doi.org/10.1016/j.jse.2015.08.032.)

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**Table 6**
**Frequency of symptoms associated with septic and aseptic bursitis**

| | Septic Bursitis | Aseptic Bursitis |
|---|---|---|
| Tenderness (%) | 88 | 36 |
| Erythema (%) | 83 | 27 |
| Warmth (%) | 84 | 56 |
| Trauma (%) | 50 | 25 |
| Fever (%) | 38 | 0 |

***Diagnostic tests***

Leukocytosis and elevated CRP are more indicative of septic rather than aseptic bursitis.[87] Imaging studies such as CT and MRI, though rarely necessary, are sensitive and may be helpful in identifying which anatomic compartment the infection is located in and can rule out other conditions such as cellulitis, myositis, and so forth.[32] Ultrasound can be used to detect bursal distension and guide bursa aspiration.[88]

While symptoms can help differentiate septic from aseptic bursitis, bursal fluid analysis is required to confirm the etiology. Purulent aspirate favors an infectious etiology.[87] In septic bursitis, fluid WBC count is typically highly elevated (>200,000 cells/$mm^3$), with neutrophil predominance. Conversely, the WBC count in aseptic bursitis is usually less than 1500 cells/$mm^3$ with lymphocyte predominance. Crystal-induced (gouty) bursitis usually shows a fluid WBC in the intermediate range of 1000 to 6000 cells/$mm^3$.[83] Although not commonly performed in practice, bursal glucose less than 31 mg/dL or less than 50% the serum glucose level also suggests septic bursitis. Gram stains can be negative in 30% of cases.[83] When possible, bursal fluid should be sent for cultures to identify the pathogen.

***Treatment***

Since the predominant pathogens are *Staphylococcus* and *Streptococcus* species, the antibiotic of choice should cover this pathogen, such as a first-generation cephalosporin or penicillinase-resistant penicillin.[83,87] In severe infections, and patients at increased risk, MRSA coverage should be provided while awaiting culture results. There is differing opinion on the need for bursal fluid aspiration prior to the start of antibiotics. Up to 88% of patients with suspected uncomplicated septic bursitis treated empirically with antibiotics without bursal aspiration had resolution of symptoms.[76] This approach may lead to decreased complications from bursal aspiration.

Patients with mild-to-moderate septic bursitis can be managed with a 2 week course of oral antibiotics.[74] Failure to reach resolution within 10 days is considered failure of antibiotic treatment.[89] Patients with severe bursitis (extensive localized infection or systemic symptoms) or immunosuppression should be admitted for intravenous antibiotics followed by oral antibiotics for a total of 2 weeks.[74] Patients with complicated septic bursitis may ultimately need bursectomy for definitive treatment.

## SUMMARY

Acute joint pain has a broad differential that includes both articular and periarticular causes of pain. Obtain a focused history that assesses for risk factors including the presence of an immunocompromise or immunosuppression, or the presence of orthopedic hardware, where the presentation can be indolent, the diagnosis more difficult and treatment course more complicated. A detailed joint examination can help

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distinguish between articular and periarticular causes. Localized swelling and tenderness is suggestive of extra-articular causes such as bursitis and tendinitis, whereas generalized joint tenderness and very painful range of motion is suggestive of intra-articular pathology. If there is clinical concern for septic arthritis, obtain synovial fluid for analysis. Interpret the results in the setting of the patient's clinical picture and associated risk factors. For suspected PJI, consult an orthopedic specialist immediately. In cases when the diagnosis is unclear, err on the side of caution and admit patients for close monitoring and further evaluation.

## CLINICS CARE POINTS

- Septic arthritis typically presents with acute monoarticular arthritis but 1 in 5 patients have more than 1 joint involved.
- The lack of systemic symptoms or laboratory findings such as fever or leukocytosis does not rule out septic arthritis.
- Patients with prosthetic joints or those who are immunocompromised may have an insidious presentation.
- *Salmonella* is the most common pathogen in patients with SLE, particularly with high-dose steroid use.
- Crystalline disease can coexist with septic arthritis. Therefore, the presence of crystals does not exclude septic arthritis.
- When there is concern for septic arthritis, arthrocentesis must be performed unless there is a strong contraindication.
- The classic triad of disseminated gonococcal infection includes migratory polyarthritis, tenosynovitis, and skin lesions. When there is a suspicion of disseminated gonococcal infection with arthritis, in addition to synovial fluid cultures, which have low yield, NAAT and culture of other mucosal sites should be obtained, along with blood cultures.
- Patients with septic bursitis typically present with localized pain, tenderness, warmth, and swelling but joint ROM is preserved.

## DISCLOSURE

The authors have nothing to disclose.

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